Conferences and Reviews

Cervical Spondylosis
An Update

BRUCE M. McCORMACK, MD, *and* PHILLIP R. WEINSTEIN, MD, *San Francisco, California*

Cervical spondylosis is caused by degenerative disc disease and usually produces intermittent neck pain in middle-aged and elderly patients. This pain usually responds to activity modification, neck immobilization, isometric exercises, and medication. Neurologic symptoms occur infrequently, usually in patients with congenital spinal stenosis. For these patients, magnetic resonance imaging is the preferred initial diagnostic study. Because involvement of neurologic structures on imaging studies may be asymptomatic, consultation with a neurologist is advised to rule out other neurologic diseases. In most cases of spondylotic radiculopathy, the results of conservative treatment are so favorable that surgical intervention is not considered unless pain persists or unless there is progressive neurologic deficit. If indicated, a surgical procedure may be done through the anterior or posterior cervical spine; results are gratifying, with long-term improvement in 70% to 80% of patients. Cervical spondylotic myelopathy is the most serious and disabling condition of this disease. Because many patients have nonprogressive minor impairment, neck immobilization is a reasonable treatment in patients presenting with minor neurologic findings or in whom an operation is contraindicated. This simple remedy will result in improvement in 30% to 50% of patients. Surgical intervention is indicated for patients presenting with severe or progressive neurologic deficits. Anterior cervical approaches are generally preferred, although there are still indications for laminectomy. Surgical results are modest, with good initial results expected in about 70% of patients. Functional outcome noticeably declines with long-term follow-up, which raises the question of whether, and how much, surgical treatment affects the natural course of the disease. Prospective randomized studies are needed to answer these questions.

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Clumsy hands and the wide-based, stooped, somewhat jerky gait of elderly persons have been described throughout the ages. Likewise, neck and upper extremity pain is such a ubiquitous ailment of aging that "pain in the neck" has entered our language as a figure of speech. It was not until 1952, however, that it was recognized that the myelopathy and radiculopathy from cervical spondylosis constituted clinical disorders.[1] Degenerative disease of the cervical spine and its cartilaginous and ligamentous structures is known to be the most common cause of cervical cord and root dysfunction in patients older than 55 years.[2] Pathogenic mechanisms have been better defined,[3-5] in part, through advances in neuroimaging, such as magnetic resonance (MR) imaging, which has also simplified diagnosis and facilitated treatment.[6-10]

Treatment was largely supportive until 1892 when Victor Horsely performed a cervical laminectomy for a patient with spondylotic myelopathy.[11] Anterior cervical spine approaches were developed in the 1950s as the mechanism of disease and the pathophysiology of radiculopathy and myelopathy were better defined.[12,13] In the 1970s, the use of the operating microscope and microsurgical techniques for cervical spine surgery was popularized.[14,15] More recently, spinal instrumentation developed for the treatment of traumatic instability[16-18] has been used in patients with cervical spondylosis to do radical decompressions, provide spinal stability, and enhance bone healing.[19]

In this article, we summarize the salient features related to the pathogenesis, clinical diagnosis, and treatment of cervical spondylosis.

Pathophysiology

Cervical spondylosis is caused by a degeneration of the intervertebral discs, which fragment, lose water content, and collapse with normal aging.[20,21] Disc degeneration causes increased mechanical stress at the cartilaginous end plates at the vertebral body lip. This results in subperiosteal bone formation or osteophytic bars that extend along the ventral aspect of the spinal canal and, in some cases, encroach on nervous tissue. Osteophytes or "hard disc disease" should be differentiated from soft disc herniations of fibrocartilage that occur in young and middle-aged adults.[1] Spondylotic changes in the cervical spine occur at solitary disc space levels in 15% to 40% of patients and at multiple levels in 60% to 85%.[1,22] The discs

From the Department of Neurological Surgery, University of California, San Francisco, School of Medicine, and the Veterans Affairs Medical Center, San Francisco. Reprint requests to Bruce M. McCormack, MD, Veterans Affairs Medical Center, Dept of Neurological Surgery (Box 112), 4150 Clement St, San Francisco, CA 94121.

ABBREVIATIONS USED IN TEXT

CT = computed tomography
EMG = electromyography
MR = magnetic resonance

between the third and seventh cervical vertebrae are affected most commonly.

Repeated occupational trauma may contribute to the development of cervical spondylosis.[1,23,24] An increased incidence has been noted in patients who carried heavy loads on their heads or shoulders, dancers, gymnasts, and in patients with spasmodic torticollis.[22] Not everyone agrees that trauma is an important causal factor in the production of this disorder.[1] In about 10% of patients, cervical spondylosis is due to congenital bony anomalies—blocked vertebrae, malformed laminae—that place undue stress on adjacent intervertebral discs.[1,22]

Cervical spondylosis may result in symptomatic spinal cord compression.[1,20-22] The presence of a congenitally narrow spinal canal—diameter of 10 to 13 mm—is a critical predisposing factor in patients with spondylotic myelopathy.[3,5,25-29] Congenital spinal stenosis is poorly understood and occurs sporadically, within families, and in achondroplastic dwarfs.[30] Symptomatic cord compression occurs as degenerative spinal changes result in further narrowing of a developmentally narrow spinal canal. Degenerative stenosis is usually caused by ventral spondylotic bars. The thickening of bone and ligaments of the spinal column, laminar "shingling," and ligamentum flavum buckling into the posterior spinal canal also contribute to spinal stenosis. Pathologic vertebral body subluxations may further narrow the sagittal diameter of the canal. Other mechanisms, aside from static cord compression, have been implicated in the pathophysiology of cervical spondylotic myelopathy.[3-5] Cervical motion causes chronic cumulative cord trauma from impaction of the spinal cord against bony spurs or from pathologic subluxation of vertebral bodies. Acute spinal cord injury may occur from neck extension when the cord is pinched between anterior osteophytes and an infolding ligamentum posteriorly. Neural ischemia from repetitive minor contusion due to trauma and vascular compromise due to stenosis are also thought to be contributory.

The spinal cords of patients with cervical spondylotic myelopathy are flattened at the levels of spondylotic protrusions.[31,32] At the level of compression, the gray matter is ischemic, with a loss of neurons and, in some cases, cavitary lesions. There is degeneration of the lateral descending columns below the level of compression; ascending posterior columns degenerate above the compressed level. White matter lesions include irregular areas of pallor, necrosis, and demyelination.

Cervical spondylotic radiculopathy is caused by nerve root compression in the neural foramina.[33-35] The root normally occupies about a third of the space in the foramen and is accompanied by radicular arteries and veins. The root is vulnerable to compression by the facet joint posteriorly or the uncovertebral joints and disc anteriorly. The facet and uncovertebral joints may hypertrophy, or the disc may rupture or become calcified. It is conjectured that nerve root irritation may also occur from a degradation of discal proteoglycans without direct compression.[36] Nerve roots in patients with spondylotic radiculopathy are flattened, with surrounding fibrosis of the root sleeve. Wallerian degeneration is uncommon.[1]

Clinical Spectrum and Natural History

Cervical spondylosis occurs in middle-aged or elderly patients. It may cause neck pain syndromes, myelopathy, or radiculopathy.[1-3,25,34,35,37] Neck pain and stiffness with radiation into the shoulders or occiput may be chronic or episodic with prolonged periods of remission. Flexion-extension injuries, blows to the head, or neck injury while lifting heavy objects may precipitate an acute exacerbation. Neck pain usually accompanies upper extremity radicular symptoms and often may be absent in patients with myelopathy.[1,22,38-41] Abnormal findings of an examination consist of decreased mobility, muscle spasms, and tenderness.

Radicular and myelopathic symptoms are usually distinct syndromes with little overlap.[42] There are fewer cases of myeloradiculopathy in which signs of nerve root dysfunction in the upper limbs accompany long tract signs in the lower limbs. In these patients, it is difficult to know for certain whether to ascribe findings in the upper limbs to a lesion of the nerve roots or to one of the corresponding segments of the spinal cord. Some think that myeloradiculopathy is the most common neurologic presentation of cervical spondylosis.[22,27]

Myelopathy

Myelopathy due to cervical spondylosis usually develops insidiously, although episodes of abrupt deterioration occur.[2,21,22,43] Acute spinal cord injury may rarely occur in elderly patients after traumatic cervical spine hyperextension.[44] The syndrome of "numb, clumsy hands" has been described in patients with high compressive myelopathy between C-3 and C-5. Typical symptoms are loss of manual dexterity, with difficulty writing; diffuse, nonspecific arm weakness; and abnormal sensations.[45,46] Lesions at levels C-5 to C-8 cause a syndrome of spasticity and proprioceptive loss in the legs. Patients have difficulty walking and an unsteady feeling; they often lose their balance and fall. Urinary frequency and urgency are common. A complete loss of bowel and bladder function is an end-stage deficit that is rare.

Neck flexion and extension may elicit electric shock sensations in the extremities (Lhermitte's sign). There is motor weakness, sensory loss, and spasticity with exaggerated reflexes below the level of spinal cord compression. Extensor plantar responses are elicited. Myelopathic syndromes may localize asymmetrically to one side of the body (Brown-Séquard syndrome). The deficit may affect predominantly motor function with preserved sensation (anterior cord syndrome) or cause hand weakness proportionally greater than leg weakness (central cord syndrome). Neurologic deficits have been graded on the basis

of gait[47]; effects on activities of daily living[48]; or motor, sensory, and bowel function.[49]

The natural history of cervical spondylotic myelopathy is variable, with some patients having a mild protracted course and others progressive disability.[22,28,39,47,50,51] In a report on 26 untreated patients, motor deficits were found to have developed over time without complete remission.[22] In 19 patients, myelopathy progressed episodically, usually with ongoing deterioration between episodes. In 5 patients, there was slow, steady progression without remission phases from the onset onward, whereas in 1 there was a rapid onset followed by lengthy periods of stability. In a review of 44 patients who had myelopathy on initial presentation, the course of the disease was noted to be "benign," with long periods of nonprogressive disability the rule and a progressively deteriorating course the exception.[39] Those who disagree with these conclusions observed that 25 of the patients in that study (57%) had severe disability at some time, and in most this remained[51]; only 8 patients (18%) improved based on the disability category.[28] In 1967 a report of the experience of 48 patients found a less favorable natural history, with 32 patients having a steadily progressive deterioration.[28] In a review of the literature, it was found that 30% to 50% of patients with myelopathy improved with nonoperative treatment.[50]

Radiculopathy

Radiculopathies develop insidiously or may be triggered by trauma.* Because more than one cervical spine segment is often affected, symptoms are more diffuse than those associated with unilateral soft disc herniation, and they may be bilateral. Acute neck and arm pain, paresthesias, and weakness are typical, but one condition may exist without the others. Less commonly, the pain may radiate to the chest or face. On examination, sensory loss, weakness, and hyporeflexia may be seen in a radicular pattern. In advanced cases, muscle wasting and fasciculations may occur. The C-6 and C-7 roots are the most commonly affected.[34,40,52,53,55] The Spurling maneuver may be done to elicit radicular symptoms. It is performed by extending the patient's neck, rotating the patient's head to the side of the pain, and then applying downward pressure on the head.[56]

A recent epidemiologic survey of cervical radiculopathy indicates that symptoms resolve in 75% of patients with conservative measures.[53] A fifth of the patients in that survey were treated surgically. At six years' follow-up, 90% of patients were asymptomatic or only mildly incapacitated. Disparate outcomes for conservative management of cervical radiculopathy have been reported. Referral center-based studies indicate persistent pain and incapacity in two thirds of patients treated conservatively.[33,39] Surgery-based studies indicate that complete relief with nonoperative therapy occurs in only 29% of patients.[57] Another study found that 23% of patients remain partially or totally disabled.[58] Physiotherapy centers find, however, that 70% to 92% of patients have good relief with physiotherapy and cervical traction.[54,55,59,60]

*References 1, 22, 33-36, 38, 40, 42, and 52-54.

Differential Diagnosis

Cervical spondylosis is ubiquitous in elderly persons, and neurologic dysfunction may or may not be attributable to spondylotic cervical spine changes seen on imaging studies.[6,61] Misdiagnosis is a well-recognized cause of a poor surgical outcome.[62] Neurologic consultation is advised to interpret clinical findings and obtain radiologic and electrophysiologic tests.

The following diseases should be considered in the differential diagnosis of cervical spondylosis with myelopathy or myeloradiculopathy: motor neuron disease, multiple sclerosis, spinal cord tumor, syringomyelia, and tropical spastic paraplegia.[62,63] Spinal cord tumor and syringomyelia are readily diagnosed with MR imaging. Tropical spastic paraplegia is attributable to human T-lymphotropic virus type I and occurs in patients from the Caribbean region and in patients infected through blood transfusions.

It may be difficult to differentiate between cervical spondylotic myeloradiculopathy and motor neuron disease. Both conditions tend to appear in older patients. The diagnosis of motor neuron disease should be favored if sensation is normal or if muscular fasciculations are present in the tongue, face, or lower extremities.[62,63] The clinical diagnosis is supported by typical findings on electromyography (EMG): substantially longer duration of the motor unit potentials combined with signs of denervation, such as fibrillations, sharp positive waves, and fasciculations.

The spinal form of multiple sclerosis may mimic the clinical course of cervical spondylotic myelopathy and radiculopathy.[34,62,63] Patients with cervical myelopathy are usually older, oligoclonal bands in the cerebrospinal fluid are absent, and the visual evoked potentials are normal. Magnetic resonance imaging may detect demyelinating plaques in patients with multiple sclerosis, but white matter lesions, probably vascular in origin, are frequently seen in patients older than 50.

The differential diagnosis for cervical radiculopathy includes upper limb nerve entrapment syndromes and brachial neuritis (neuralgic shoulder amyotrophy).[34,63] The carpal tunnel syndrome and ulnar neuropathy may be differentiated from radiculopathy because symptoms are in the distribution of a single peripheral nerve whereas multiple nerves are involved with radiculopathy. Median neuropathy at the wrist, however, may present with proximal symptoms as high as the neck. Tinel's sign, which consists of radiating paresthesia in the distribution of a peripheral nerve when the nerve is tapped with a finger, is often present with nerve entrapment. In some patients, the diseases may coexist, referred to as the double-crush syndrome.[64,65] The syndrome occurs from the proximal compression of the nerve root at the neural foramen, which weakens the nerve's ability to withstand distal compression at the wrist or elbow. Electromyography will distinguish between an entrapment syndrome and radiculopathy and, if they are both present, define the distribution of abnormalities.

Acute brachial radiculitis refers to acute shoulder and neck pain followed by weakness and atrophy of the shoul-

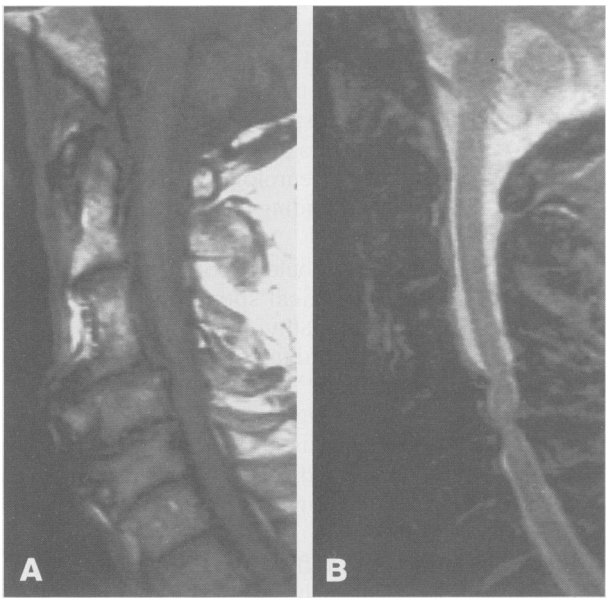

Figure 1.—A, A sagittal T1-weighted magnetic resonance (MR) image of the cervical spine in a patient with myelopathy shows detailed gross anatomy. Cord compression is seen at C3-4 and C4-5. **B,** Spin echo or gradient echo T2-weighted MR images provide a "central nervous system myelographic" effect to highlight anterior cord compression. Increased signal intensity in the cord on this T2-weighted image indicates cord damage and may be a poor prognostic sign.

der girdle and the upper limb muscles due to an inflammatory allergy. Symptoms are bilateral in 25% of cases.[63,66-68] The syndrome may be differentiated from cervical spondylotic radiculopathy by the high intensity of the pain followed by weakness, after which the pain usually recedes, the absence of a Spurling's sign, normal findings on a cervical paraspinal EMG examination, and imaging studies that do not show a lesion sufficient to result in a severe neurologic deficit.

Other diseases may affect the cervical spine and cause spinal cord or root impingement. Rheumatoid arthritis affects the cervical spine in 36% to 88% of cases.[69] Myelopathy may result from basilar invagination, C1-2 subluxation, and spondylolisthesis of other cervical vertebrae. Radiculopathy may result from osteoarthritis of intervertebral and uncovertebral joints. Ossification of the posterior longitudinal ligament, ankylosing spondylitis, and diffuse idiopathic skeletal hyperostosis are arthritic conditions that may result in neurologic manifestations identical to cervical spondylosis.[70]

Imaging Studies

Plain radiographs are an inexpensive initial diagnostic study. They are of limited specificity because degenerative changes are present in both symptomatic and asymptomatic patients.[61] A loss of disc space height and osteophyte formation with narrowing of the sagittal canal diameter are visualized on the lateral projection. A narrow spinal canal, with a sagittal diameter of 10 to 13 mm, has been associated with a higher incidence of neurologic deficits,[5,25-29] but this measurement is less important with the introduction of MR imaging, which directly visualizes neural structures. Neural foraminal narrowing is seen on oblique views. Plain radiographs are helpful in assessing spinal alignment and the contribution of degenerative spondylolisthesis—relative displacement of one vertebral body to another—to canal stenosis. Cervical spine flexion and extension views are used to assess spinal stability.

Magnetic resonance imaging is the preferred initial imaging study (Figures 1 and 2).[71-73] In one study, MR imaging correctly identified 88% of the surgically proved lesions, compared with 81% for postmyelographic computed tomography (CT), 58% for myelography, and 50% for CT.[72] Advantages include the lack of irradiation, the avoidance of invasive intrathecal contrast administration, and the capability of multiplanar imaging.[71] In addition to its greater sensitivity in the detection of disc disease and extradural compression, MR imaging also better displays

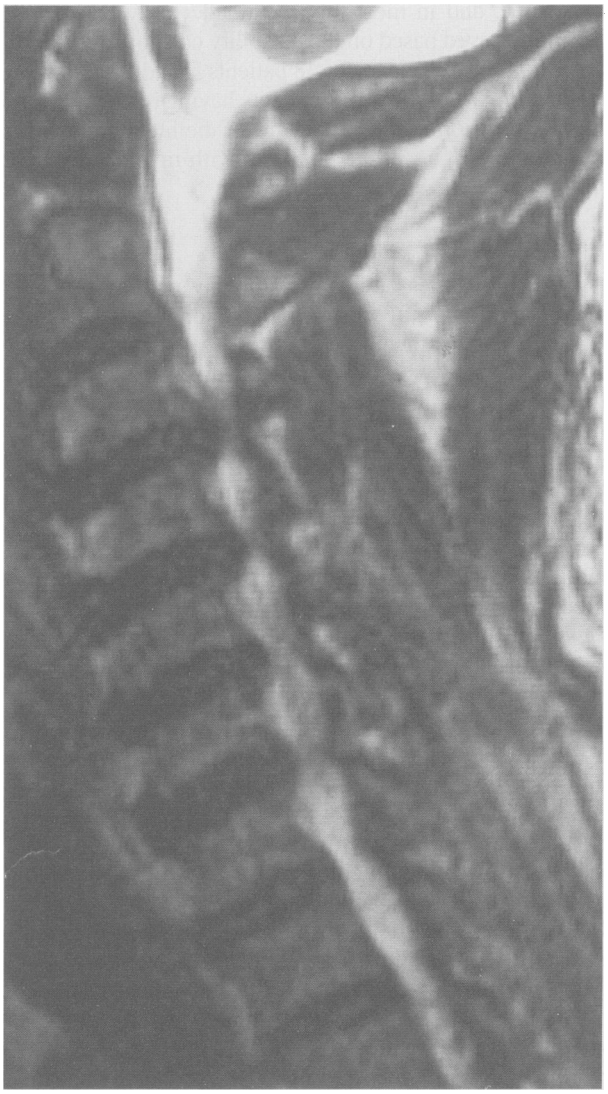

Figure 2.—A sagittal T2-weighted magnetic resonance image is shown of a patient with myelopathy. The spinal canal is developmentally narrow with cord compression at every level. This patient was treated with laminectomy.

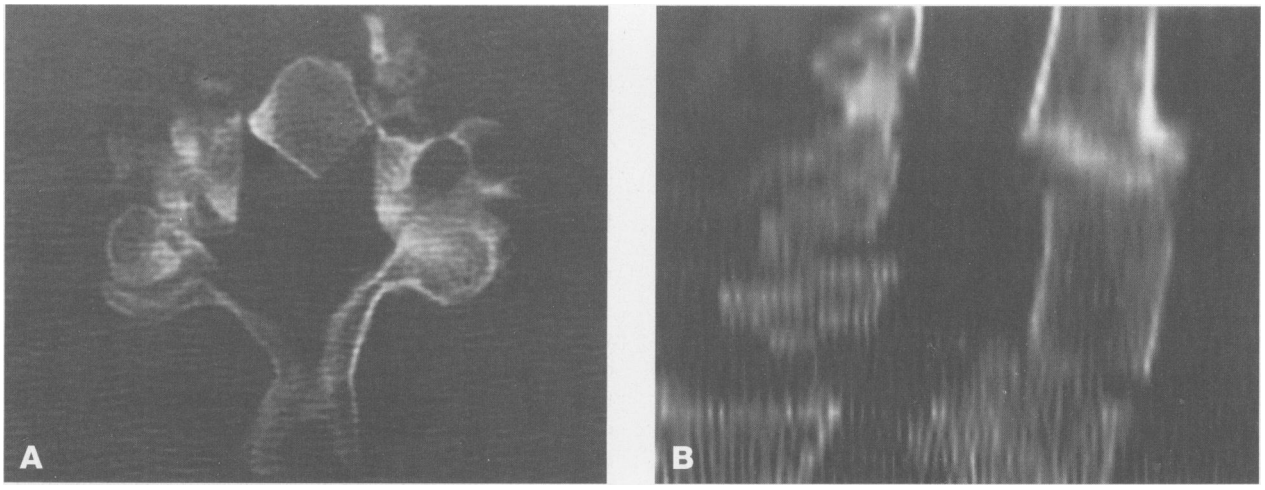

Figure 3.—A, A computed tomographic (CT) scan with sagittal reconstruction is shown in the same patient as in Figure 1 after C-4 vertebrectomy. **B,** An axial CT scan shows wide decompression of the canal with a bone plug in good position. Note the exuberant bony overgrowth of anterior osteophytes.

intramedullary cord disease.[7] A limitation of MR imaging is that it may not provide enough bone detail.[71] Cervical MR imaging is also subject to more artifact and distortion than is lumbar MR imaging.

Computed tomography after the introduction of water-soluble contrast agents is an alternative accurate method for evaluating patients with neurologic symptoms.[71] Many surgeons prefer CT myelography for the evaluation of radiculopathy because CT provides superior imaging of bone compared with MR imaging and better defines the anatomy of the neural foramina. Computed tomography is often used to complement MR imaging to provide additional bony detail to characterize the lesion responsible for neural entrapment (Figure 3).[72]

Treatment

The treatment of cervical spondylosis may be medical or surgical, depending on whether a patient presents with symptoms of myelopathy, radicular pain, or neck pain. These clinical symptoms overlap, but are discussed separately for clarity.

Myelopathy

Patients presenting with mild myelopathic symptoms may be treated conservatively and observed over time because many such patients have a protracted course of minor impairments without progression.[22,39,47,50] The cornerstone of conservative therapy is to immobilize the cervical spine with a collar that holds the head in a neutral or slightly flexed position.

Surgical treatment is recommended for patients with moderate or severe disability when first seen because conservative therapy yields an improvement rate of only 30% to 50% of such patients.[50] The effects of surgical treatment on the natural history of spondylotic myelopathy have been questioned,[39,41,62,74] and prospective, randomized studies have been proposed to clarify the role of surgical therapy.[41,62,75] Despite some controversy, the bulk of evidence suggests that patients with moderate to severe symptoms of myelopathy are best treated surgically. Several clinical and radiologic criteria have been recognized as possible predictors of outcome, including abnormal spinal cord signals on MR imaging that may portend a poor prognosis (see Figure 1-B).[7,9,10,38,41,76] In our experience, a brief duration of symptoms and mild neurologic deficits are associated with a good outcome. Long-standing neurologic disability and traumatic spinal cord injuries are poor prognostic factors. The best that can be anticipated in such cases is to prevent further deterioration and perhaps slightly improve gait and hand function.

For patients with spondylosis who have acute spinal contusion from neck hyperextension,[44] emergency medical treatment consists of administering methylprednisolone sodium succinate, 30 mg per kg of body weight in a bolus, followed by 5.4 mg per kg per hour for 23 hours, to be started within eight hours of injury. In a recent study, patients treated with steroids were neurologically improved at six months' follow-up compared with patients treated with placebo.[77] If indicated, surgical intervention is ideally deferred until spinal cord swelling has resolved and the neurologic state has become stable.

Surgical therapy for spondylotic myelopathy may be through either an anterior or a posterior approach; several large series have failed to establish the superiority of either procedure (Figure 4).[49,76,78-80] Myelopathy caused by osteophytes confined to one or two levels is treated by an anterior operation with the removal of osteophytes.[38,41,51,81,82] In severe cases, radical anterior surgical decompressions are done using multiple-level vertebrectomies and reconstruction with instrumentation.[19] Indications for posterior decompression (that is, laminectomy) are encountered less frequently and include cord compression from posterior structures (that is, ligamentum flavum and hypertrophied facet or laminar bone) and the presence of a developmentally narrow spinal canal (see Figure 2). The treatment of multilevel disease is controversial. Many surgeons prefer laminectomy for multilevel disease,[76,78,83-85] whereas we prefer to treat these patients with an anterior approach. Laminectomy may re-

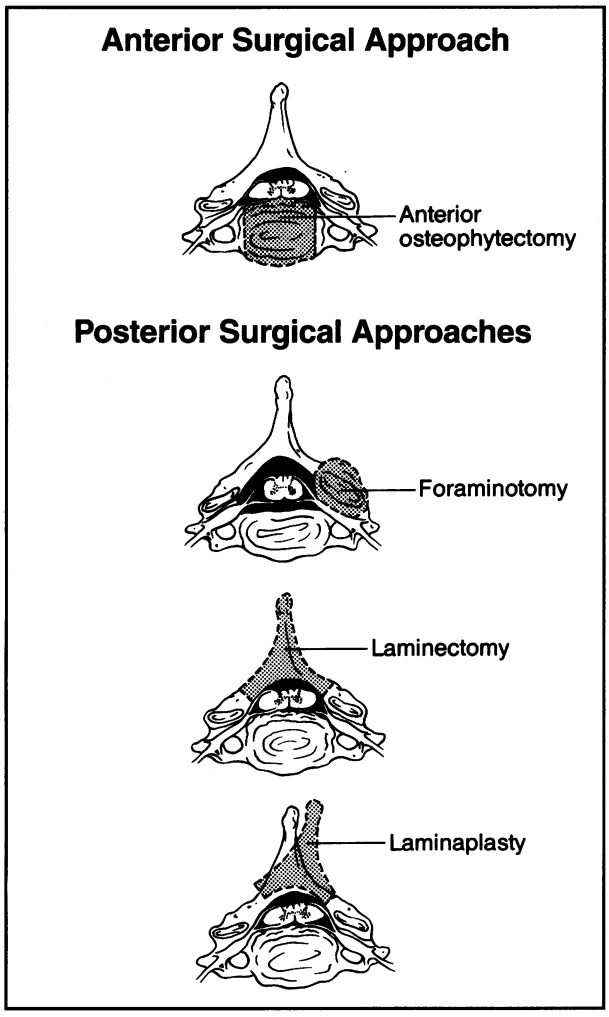

Figure 4.—The anterior and posterior approaches to the cervical cord and nerve roots are depicted. Three posterior procedures are illustrated: laminectomy, foraminotomy, and laminaplasty. Laminaplasty procedures expand the spinal canal by partially removing the lamina and elevating the remaining bone fragments.

sult in spinal instability in as much as 10% of patients and is contraindicated if there is cervical spine kyphosis.[85,86] Laminaplasty, a technique to enlarge the spinal canal by preserving and elevating the lamina roof over the dura, may to some extent prevent spinal instability.[49,87-89]

Recent surgical series using both anterior and posterior approaches show excellent or good results in about 70% of patients with myelopathy.[37,38,49,90-95] Fewer studies report less encouraging results.[41,47,62] In general, results are better with an anterior operation, with improvement reported in 70% to 80% of patients.[49,95-100] Even more impressive results have been reported with radical anterior decompressions (vertebrectomy).[19,81,101-105] The results of an anterior procedure may, in part, be biased favorably because usually the extent of the disease has been limited to one or two levels and follow-up has been short.[76] Misdiagnosis, surgical trauma, inadequate decompression, and irreversible spinal cord injury are important identifiable reasons for a poor surgical outcome.[106-109] At long-term follow-up, functional outcome noticeably declines, but the decline is clearly more pronounced in those patients with a posterior operation.[38,76,91,94,109,110] Delayed neurologic deterioration may occur from disease progression at other levels and spinal instability.[109] About 20% of patients deteriorate without radiologic explanation,[76,106,107] indicating that the pathophysiology of cervical spondylotic myelopathy is still enigmatic.

Cervical Radiculopathy

Radicular symptoms resolve in the majority of patients with simple remedies[34] or without any treatment whatsoever.[39] Treatments include activity modification, neck immobilization, intermittent cervical traction, and isometric exercises when acute neck pain resolves. Nonsteroidal anti-inflammatory agents will usually provide adequate pain relief. Diazepam and narcotics are used sparingly. A short course of steroids—prednisone, 60 mg daily for 7 days and then 5 days tapered—is given occasionally for severe cases. The instillation of steroids into the cervical epidural space may be of benefit in patients with pain that does not resolve with the above therapy.[34]

The indications for surgical intervention are unremitting pain and progressive weakness despite a full trial of nonsurgical management. Anterior and posterior approaches have been used to perform root decompression, with equal results.[33,111] Foraminotomy, or a posterior approach, is preferred for nerve root compression due to facet joint hypertrophy and may be preferred for posterolateral disc herniations. The anterior approach is more appropriate if root compression occurs from osteophyte or disc located anterior to the root (see Figure 4). If an anterior operation is elected, the removal of disc and osteophyte is usually followed by the placement of an interbody strut graft harvested from the hip or from a bone bank. Others prefer not to use a strut graft and claim that perioperative morbidity is reduced, complications of grafting are avoided, and overall clinical results are comparable.[40,112-116] We think that grafting maintains spinal anatomic alignment and neural foraminal patency and maximizes the chance for solid bony fusion. Without a graft, collapse at the discectomy site can cause prolonged postoperative neck pain and narrow the neural foramen at that level, with the potential for the development of a radiculopathy. Instrumentation improves bony fusion rates, but because instrumentation may result in complications, it is generally not warranted in routine cases.

Excellent or good results have been reported in 70% to 80% of patients after surgical treatment of spondylotic radiculopathy using either anterior or posterior approaches.[38,40,42,83] In one study, one or more symptoms recurred in 60% of patients within one to six years after the operation, but episodes of deterioration responded to conservative therapy and rarely required further surgical intervention.[40]

Neck Pain

Neck pain from spondylosis, in the absence of radicular symptoms, will usually respond to conservative therapy as outlined for patients with radiculopathy. Depressed

persons, those using disability for secondary gain, and patients with myofascial pain disorders should be identified. Surgical treatment is not advocated for neck pain from spondylosis.[33] In our experience in treating patients with radiculopathy, localized neck pain from single-level disc disease may resolve with surgical treatment, but pain from multilevel disease responds less predictably.

Summary

Neck pain from cervical spondylosis will usually respond to simple remedies, including activity modification, neck immobilization, isometric exercises, and drugs. For patients with neurologic symptoms, MR imaging is the preferred initial diagnostic study, although CT myelography may still be preferred for evaluating radiculopathy. In most cases of spondylotic radiculopathy, symptoms resolve with conservative therapy, and surgical intervention is not considered unless pain persists or there is progressive neurologic deficit. When indicated, surgical therapy is performed through either the anterior or posterior cervical spine, depending on the site of neural impingement. Surgical results are gratifying, with improvement in 70% to 80% of patients. Myelopathy is the most severe and disabling feature of this disease. The natural history of spondylotic myelopathy is variable, with some patients having nonprogressive minor impairment and others relentless neurologic deterioration. Because the clinical course in any new patient is unpredictable, it is reasonable to treat patients who have minor symptoms with a collar and observe them over time. A third to half of patients improve with this simple remedy. An operation is advocated for moderate or severe disability. In general, anterior cervical approaches are preferred, although there are still indications for laminectomy. Results are modest, with initial satisfactory results expected in 70% of patients. Functional outcome declines with long-term follow-up, but the decline is clearly more pronounced in those patients with a posterior operation. This finding raises the question of whether, and how much, surgical treatment affects the natural course of the disease. Prospective randomized studies will answer these questions.

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